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The Journal of HEREDITY

A MEMBER OF THE BROKEN-BONE FAMILY

During childhood their long arms and legs fractured many times.

VOLUME 22 APRIL, 1931 NUMBER 4

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

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CROOKED ARMS AND LEGS DUE TO MANY BENDS AND FRACTURES

Frontispiece

When this woman was a baby she broke her leg by getting tangled in a blanket—and later broke it again playing with other children on the floor. She had many other fractures as she grew up. Her uncle fractured his legs on an average once a year until he was sixteen. All the broken-bone people have very long arms and legs and are of rather short stature. This form of bone fragility is inherited as a Mendelian dominant character.

A FRAGILE-BONED FAMILY

Hereditary Fragilitas Ossium

D. Y. Holcomb
University of Arkansas

CASES of hereditary bone fragility have been studied and described for more than a hundred years, though fortunately it is a relatively rare disease. In the earliest papers no mention was made of the various other abnormalities which are now known to accompany the excessive liability to bone fracture, but in 1896, Spurway[19] published an account of a family in which the hereditary tendency to fracture extended through four generations, and noted the fact that a curious bluish tinge of the eyeball was common to many members of the family. Four years later Eddowes,[6] reported several cases which had come under his observation and also pointed out the peculiar fact that blue sclerotics and osseous fragility often occur in the same individual. Since that time it has been found that a large number of the persons with brittle bones have blue sclera, and in more recent years several investigators have discovered that deafness is also very commonly found in connection with the bluish coloring of the sclerotics and the fragilitas ossium. This deafness usually appears in early adult life and tends to increase with age.

In the Davis family, whose history I have investigated, the tendency to break bones with abnormal frequency, especially in childhood, occurred in a number of persons in five generations of the family, but in no case was there the slightest indication of the blue sclera or progressive deafness. There are, however, other less striking conditions such as shortness of stature, abnormally rapid healing of broken bones and cessation of fractures at puberty, which have been found to be frequently associated with bone fragility. Most of these peculiarities are apparent in the affected members of the present family and will be discussed later.

Causes Not Known

Although fragilitas ossium has been known for so many years and numerous cases have been recorded in the extensive literature on the subject, there is no agreement as to its pathology, and there seems to be no general conformity in the terminology used. Key[13] who has recently made a very careful study of the subject states that "It is one of the most obscure subjects in modern medicine." The cases recorded in this paper may be classified as osteopsathyrosis idiopathica. According to Scott[17] this term was originated about 1833 by Lobstein who described the disease in cases occurring in infants and young children, and was the first to point out its hereditary tendency.

As to the cause of the bone fragility there is much difference of opinion. As Haas[10] states, "the fundamental explanation is to be sought in the organs that control the physiological and chemical processes in bone growth." Many authors have suggested that disturbances of the ductless glands may account, to some extent at least, for the condition. Thyroid extract and other endocrine preparations have, however, been used for treatment without successful results. Stobie[21] felt that even though there was no definite evidence that an endocrine imbalance was responsible in the family that he studied, appearance of the patients constantly suggested the idea. So in the case of the Davis family, their small stature, their peculiar facial expression, the pointed

FATHER AND SON BOTH FRAGILE-BONED

Figure 1

The father (at right, II-3 in the chart) is now eighty. He had many fractures when he was a child, which has left him somewhat deformed, but he is still quite active. The son (III-3 in the chart) broke on the average about an arm or a leg a year until he was sixteen. It is characteristic of this condition that at maturity the bones become less brittle, and fractures occur with no more than ordinary frequency.

chin, the shape of the skull and other characteristics, indicate a possible lack of harmony of the endocrines. A careful study of these cases might lead to the discovery of an inherited glandular abnormality.

There is a possibility that the peculiar bone fragility in this family may be due to the habitual lack of some vitamin in the diet, or to some other form of nutritional insufficiency. This, however, seems improbable for a number of reasons. Many members of the family are normal and the affected individuals are engaged in various occupations and live under widely different environmental conditions, so that their eating habits must vary greatly. The fact too, that the disease reappears in several generations also renders it unlikely that it is due to improper nutrition. At least it seems certain that the disease in this family is not rachitic in nature since none of the persons with the abnormal tendency to bone fracture show any of the other characteristics of rickets.

Key[13] uses the term "hereditary hypoplasia of the mesenchyme" to describe the condition of brittle bones and blue sclera, because many of the mesenchymal tissues may be underdeveloped in these cases. There is no history of sprains or dislocations in this family so far as could be discovered and since blue sclera and deafness do not appear, it is doubtful if any of the mesenchymal tissues, other than the bones, are involved.

Family History

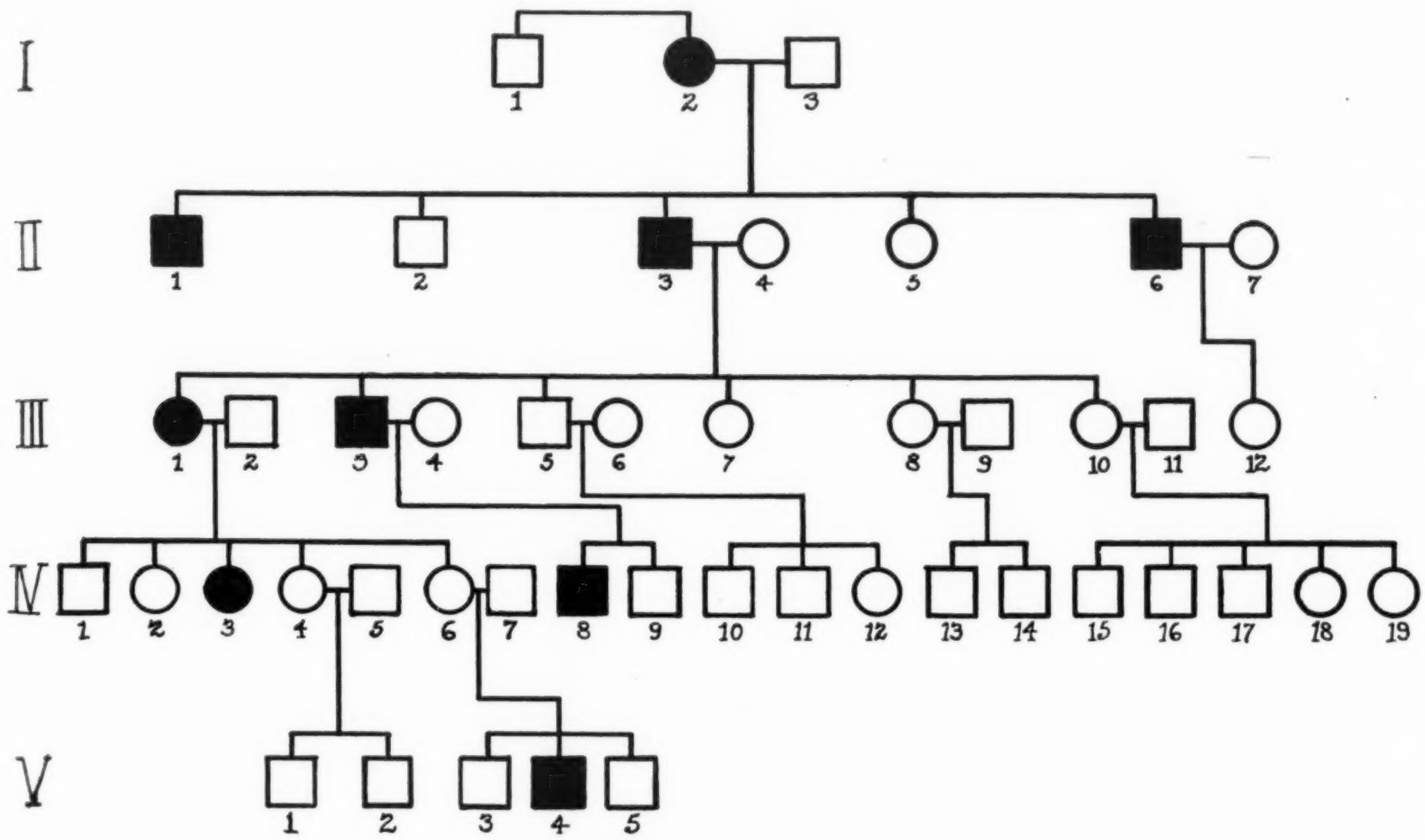
The first member of the family in whom the disease is known to have occurred was a woman who died about eighteen years ago at the age of ninety-four. Little could be learned of her history except that she had told her children that in youth she had had many fractures. She is remembered by many people as a much bent, dwarfish, little woman, who during the latter years of her life was so crippled that she was unable to walk. As shown in the accompanying chart, three of her sons inherited the abnormality.

The oldest son, II-1, had sixteen fractures. The first of these occurred when he was about one year old. As he crawled on the floor he caught his leg under a chair and broke the femur. He sustained other fractures at intervals until he was about fifteen years old; only the long bones were broken and always from trivial accidents. After puberty there were no further breaks but he was so crippled that he walked with crutches from the time of his boyhood. He lived, however, to be seventy-five years old. He had no children. The second son II-2, was unaffected. He had three daughters and two sons, and numerous grandchildren and great grandchildren, but none of his descendants show any evidence whatever of bone fragility. A brother of the two men just described, II-3, is still living and is now eighty years old. He has had numerous fractures, the first being a break of the left femur which came about as the result of a slight fall when he was four years old. Later the right femur was broken three times, the humerus of the left arm twice, and both arms were broken below the elbow. All of the fractures were in the long bones. Since there were few physicians at that time in the rural community in which he lived, the bones were usually not set. In spite of this neglect the breaks healed rapidly and with little pain, and now at the age of eighty he is still quite active though many of his bones are crooked and bent. As in the case of his brother the fractures ceased to occur after he was sixteen years old. A picture of this man is shown in Figure 1. The third affected son, II-6, has had fewer fractures than his brothers, but his crippled condition and deformed appearance offer undoubted proof that he has inherited the same defect. He

PELVIC AND LEG BONES ABNORMAL

Figure 2

The bowed femur and the asymmetrical pelvis are due to this condition. The light areas near the neck of the femur and in the pelvis indicate a defect in bone formation.



FIVE GENERATIONS OF FRAGILE BONES

Figure 3

Squares indicate males; circles, females. Theoretically half of the children of a "broken-boner" should inherit the defect. The actual ratio is 10 affected; 14 normal among the progeny of those showing the defect, including among the affected the normal individual shown in Generation IV who has an affected child (IV-6 and V-4). This exception to expectation is probably to be accounted for as due to occasional incomplete dominance of the condition.

has one daughter, and she is normal.

In the third generation, III-1, is evidently affected with the disease although she has had no fractures. At this time she is about fifty-five years old. She is a small woman, badly deformed with many twisted, misshapen bones. The family history is repeated in her brother, III-3. He fell from a small rolling log when he was three years old and broke his right femur. Later he suffered fractures of the left femur and of other bones of the legs. In fact, during his childhood bones were broken at intervals of about a year until he was sixteen years old. His general appearance, as shown in Figure 1, is typical of the affected members of the family. His arms are disproportionately long and the bones of his legs are twisted so that he walks with a decided limp, turning his left foot toward the median line with each step. The brothers and sisters of these last two persons are entirely normal and none of them are below average height and size.

In the fourth generation the most marked case of the disease is that of IV-3, a young woman now twenty-eight years old. When a baby six weeks old she became entangled in a blanket wrapped around her and broke her femur. At the age of three she broke the same femur again while playing quietly with other children on the floor. Other bones of the legs were broken a number of times, leaving her in a badly crippled condition. Her peculiar appearance is shown in the Frontispiece. She is confined to a wheel chair and with her extremely long arms is able to move about with much agility. She has had no fractures since she was twelve years old.

In the fifth generation of this family I could discover only one case, that of a boy ten years old, V-4. He has had four fractures, all confined to the bones of the legs. At one time a stick broke while he was leaning upon it, and as he pitched forward the femur was broken. The other breaks occurred as a result of equally slight accidents. Already, even though he is still a small child, he has assumed the strange appearance which characterizes the other affected members of the family.

Description of Roentgenograms

The roentgenograms show parts of the skeleton of III-3, whose appearance has already been described. He is forty-three years old, weighs about a hundred and fifteen pounds and is five feet, one inch in height. Dr. D. A. Rhinehart has examined the films and the following is quoted from his report:

Figure 2 shows the pelvis, left hip, and upper part of left femur. There is a rather marked deformity of the pelvis. The bones seem normally developed and ossified except for part of the ala of the left ilium. The central portion of this ala seems to be underossified, leaving an artificial semilunar shaped defect in the bone. This is surrounded on all sides by a rim of thick bone about 2 cms. in thickness. On the medial and posterior side, next to the sacroiliac joint, this is about twice as thick.

The head, neck and trochanteric regions of the left femur seem normal in contour. Just distal to the lesser trochanter there is a marked bowing of the femoral shaft in a lateral direction. The usual angle between the neck and shaft of the femur is replaced by a curve. Extending from about midway of the neck to a point two inches below the lesser trochanter, there is a radiolucent area in the bone. On the margins of this the cortex is thinned. The trabeculae in the bone toward the greater trochanter and at the proximal end of this defect are considerably coarsened. In the neck proximal to the defect these trabeculae cross each other forming an interlacing pattern. The defect is described as due to a deficient ossification of the cortex of the bone and of the cancellous structure of the upper extremity.

Figure 4 shows a semilateral exposure of the inferior extremity and a part of the shaft of the femur. It shows a lateral and anterior bowing of the shaft of the femur; the lateral bowing seems most pronounced at the junction of the upper and middle thirds of the shaft; the anterior bowing is uniform. The compact bone of the shaft of the femur is considerably thickened along the lateral aspect of the lateral bend. At other places the thickness of the compact bone is fairly uniform. The marrow cavity does not present irregularities. The distal extremity seems normally developed.

Figure 5 is a film of the tibia and fibula, showing the middle extent, with extremities not shown. This is a lateral view probably from the medial to the lateral projection. This film shows an anterior bowing of the tibia. This is nearly uniform, there being but a slight accentuation in the curve at the junction of the middle and distal thirds of the bone. There are apparently no irregularities in the ossification of the tibia. The fibula is quite irregular. From the proximal articulation the bone extends distally parallel with the tibia and in a normal posterior position for about three inches. The thickness and structure of the bone in this part of its extent is approximately normal. From a point three inches distal to the head, the fibula extends forward in a sweeping curve to the middle of the leg, where its anterior margin reached the level of the shin of the tibia. This portion of the bone presents a fusiform enlargement, gradually increasing in thickness toward the inferior

PECULIARITES OF THE LEG-BONES

Figure 4 **Figure 5**

At left the head of the femur and at right the fibula and tibia of III-3. The shaft of the femur is bowed. The tibia is bowed forward, and the fibula is bent forward in a sweeping curve that brings it in line with the shin of the fibula.

extent of the bone. The enlargement is caused by an increase in the diameter of the marrow cavity and by an increase in the thickness of the compact bone of the shaft both anterior and posterior to the marrow cavity. In a lateral projection of the bones of the leg the fibula should be on a level posterior to that of the tibia, except at the two extremities.

Figure 6 shows the thorax, a posteroanterior projection. This film shows the bony thoracic cage above the level of the diaphragm. From their vertebral attachments on the right side the ribs have a normal inclination downward proximal to the angles. The angles are somewhat more acute than normal. On the left side from the vertebral attachments, the ribs at first have a slight upward inclination. The angles are more acute than on the right side. From the angles forward the slope of the ribs is more pronounced than on the right side. The anterior ends of the ribs on opposite sides are at approximately the same level. The upward curve of the left ribs makes the left side of the chest higher than the right, with the apex of the left lung higher than the right and an elevation of the right shoulder. The latter is apparent in the photograph.

At the level of the seventh ribs on both sides there is a dipping inward of the thoracic wall. This is only apparent along the lateral margins of the thorax, but it probably extends entirely around the chest. Below this the ribs flare out with an increase in the thoracic diameter considerably greater in proportion than that of the upper portion. This indentation and flaring probably represents a Harrison's groove, a common finding in rachitic thoraces. Enlargements of the anterior ends of the ribs, forming a rachitic rosary, are not apparent from the film. The lungs, heart, and vascular shadows appear normal.

I regret very much that I do not have roentgenograms of the skull. The external appearance of the head, however, of III-3, suggests a probable overlapping of the parietal and frontal bones. A thick ridge extending along the area which marks the union of these bones is very noticeable. The parietal bones also are slightly more protuberant than is normal. Stobie[21], Shugrue, Rockwood and Anderson[18] and others have mentioned the prominence of these bones in cases which they have studied, and Apert[1], states that overlapping skull bones is very characteristic of osteopsathyrosis.

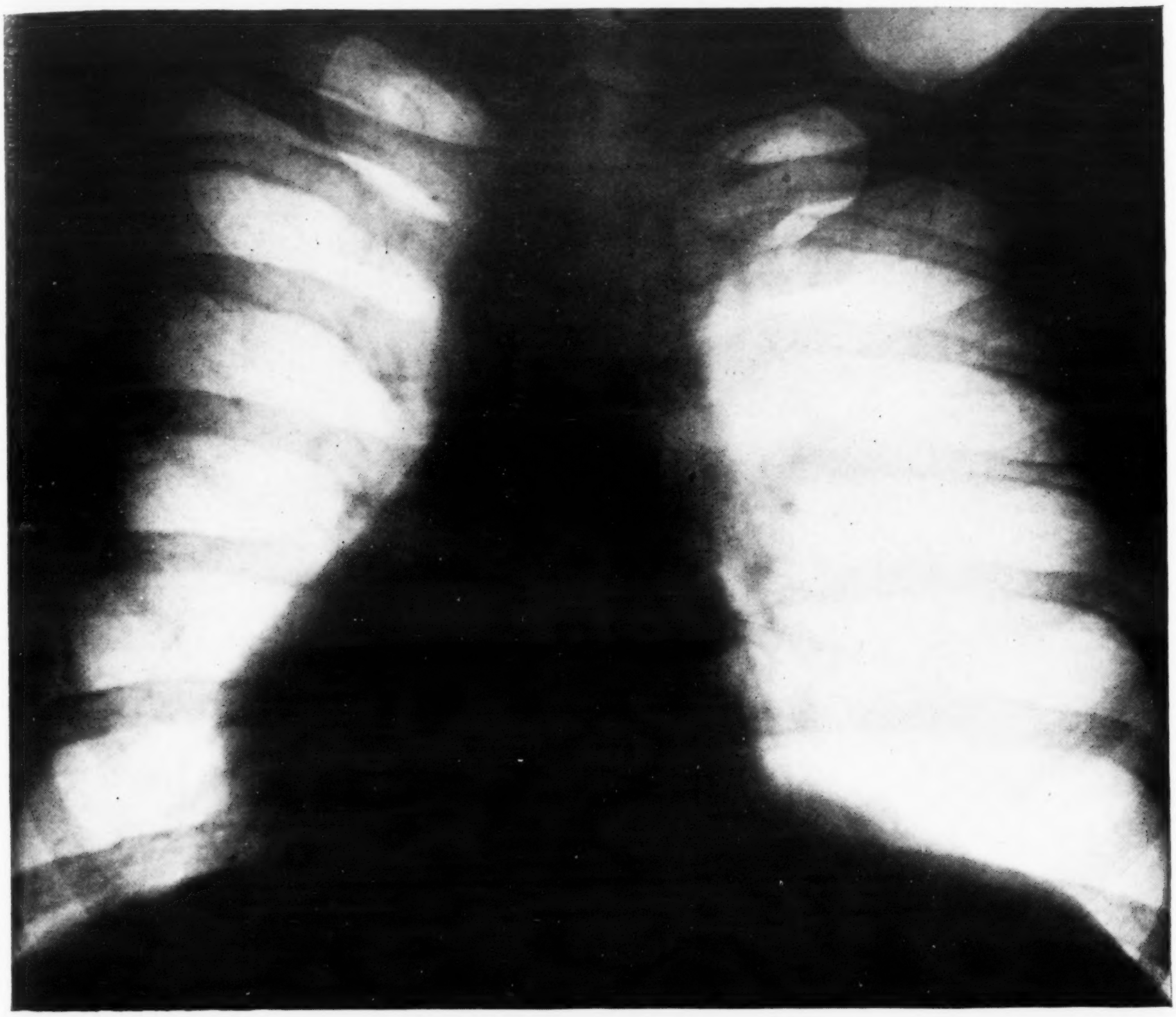
It would have been most advantageous, also, to have roentgenograms of bones of other osteopsathyrotic members of the family to use for comparison, but these could not be obtained because several of the persons concerned bitterly opposed the investigation and refused their cooperation.

Comment

As stated earlier in this paper the abnormalities found in the affected members of this family agree in the main with the conditions met with in typical cases of osteopsathyrosis except for the absence of blue sclera and progressive deafness.

The persons afflicted with the disease in the Davis family are all much shorter in stature than those individuals in the same family who are normal. The unaffected brothers and sisters are, for the most part, large and robust, with good physique. The small size of persons with osseous fragility has been mentioned by Hass[12], Harman [11], Cockayne[3], and others.

The fractures usually occur only in childhood, and the fragility of the bones seems to decrease after puberty; this agrees with the reports of Stobie[21] and Glover[8]. Bones are sometimes broken very early in life. In one case I found that the humerus was broken at birth; in another the femur was fractured at six weeks. While the breaks are fairly frequent during childhood it seems that the



RIBS BENT UP ON ONE SIDE

Figure 6

The ribs of the right are normally inclined. Those on the left are, however, inclined upward, thus making the left lung (light area) and the left shoulder higher than the right.

osteopsathyrotic individuals show no more liability to fracture bones in adult life than the average person does. Apert[1], in a recent article, described these unfortunate persons afflicted with brittle bones as "men of glass" because of the ease with which their bones are fractured. Even muscular strain may at times cause a break. One old man in the family, II-3, said that when he was a boy his thigh bone broke suddenly as he carried a bucket of water up a hill. He was quite sure that he heard the bone break, and that the fall which he sustained was the result, not the cause of the fracture of the bone. At another time he caught his foot on a briar out in the field and in jerking it loose, broke the tibia. These are fair examples of the trivial accidents which usually caused the injuries to the bones.

Many observers have noted that fractures of the long bones are the most common, and Key[13], states that European investigators believe that the femur is the bone most frequent-

ly broken. In this family practically all the fractures are confined to the long bones, and there is no doubt that the femur is fractured more often than any other bone. In fact, in several persons the same femur was repeatedly broken at different places.

The family physician who has attended three generations of the family, states that the fractures heal much more rapidly than in normal individuals. This is in accordance with results reported by Spurway[19], Key[13], and Apert[1]. Although the healing is prompt nearly all of the affected persons in this family are badly deformed. This seems to be due, in part at least, to a bowing of the long bones. This curving seems not to be uncommon in osteopsathyrosis. Glover[8] says that "the extent of deformity usually depends on the number of fractures previously sustained, although there is often an intrinsic bowing of the long bones, entirely independent of traumatic deformity". Scott[17] states that the bowing is the result of stress or strain in moving about and is most marked in legs and forearms. Apert[1], describes the fibula as appearing sometimes like a slightly curved pencil. A crippled condition, as a result of bone fragility, is not unusual. Ostheimer[16], states that "in spite of the best care, fractures recur without much cause, and over one-half of all patients remain badly crippled as a result."

Osteopsathyrosis has often been reported as associated with various other diseases, especially hemophilia, rickets, and osteomalacia. I found little evidence of any other pathological condition, though possibly the unsymmetrical pelvis shown in the roentgenogram, Figure 2, might indicate an osteomalacic condition. The bowing of the long bones, mentioned above is, in the case of this family, apparently not in any way related to the curving often found in rickets; it seems to occur only in the diaphyses and does not involve the epiphyses as would be expected in a rachitic condition.

As in other cases reported the disease does not seem to affect the longevity of the individual, since several affected members of the family have lived to be more than seventy-five years old, and none of them have died at an early age so far as I could discover.

All of the affected persons seem to be normally intelligent, though they are very ignorant and superstitious.

Heredity

In this family as in the case of many other families in which the inheritance has been studied, the factor which determines the development of the bone fragility is apparently a Mendelian dominant. The inheritance is direct from parent to child, and does not skip a generation. In one case only was there an exception to this rule. In the fifth generation a boy, V-4, evidently has inherited the abnormality from an affected grandmother through his mother who seems entirely normal. The mother is extremely sensitive about her child's condition and her attitude was so persistently hostile that I could get no information whatever from her. She has, however, had no fractures, and so far as I could tell she shows none of the stigmata of the disease. The number of affected individuals is much less than the fifty percent that would be predicted as the result of the mating of a heterozygous dominant with a normal. In the second generation in which the disease is known to occur, three of the five children of an affected mother display the bone fragility, but in the later generations the trait is transmitted to a much smaller percent of individuals. The reoccurence of the disease throughout the family is prevented to some extent by the fact that those persons in whom the abnormality is most pronounced and

who, as a result, are noticeably deformed, do not marry and have children.

The condition is transmitted apparently with equal frequency by either the mother or the father, but more males than females are affected with the disease. Conrad and Davenport[5], in their study of hereditary fragility of bone suggest that "the fact that in different families the conditions of the osteopsathyrosis are different indicates the presence of special family tendencies", and states that this "constitutes strong additional evidence of the hereditary basis of the trouble." In this family the frequency with which the femur is fractured in every affected member of the family is a noticeable peculiarity. Also, as has been previously mentioned, the course of the disease and its effects upon the different individuals are markedly similar in all the affected persons.

I wish to acknowledge my indebtedness to Dr. D. A. Rhinehart for his study of the roentgenographic appearance of the bones, and to Dr. F. B. Young for much information on the family history.

Literature Cited

1. Apert, E. Les hommes de verre; fragilité osseuse heredofamiliale avec crane à rebord, sclerotique bleues et troubles auditifs. *La Presse Medicale* 36:805-808, June 27, 1928.

2. Bronson, E. Fragilitas Ossium. *Edinburgh M. J.* 18:240. April, 1917.

3. Cockayne, D. M. Hereditary Blue Sclerotics and Brittle Bones. *The Ophthalomoscope.* Vol. XII, No. 5. May, 1914.

4. Conlon. Five Generations of Blue Sclerotics and Associated Osteoporosis. *Boston Medical and Surgical Journal,* Vol. 149, No. 1, July 3, 1913.

5. Conrad, H. S, and Davenport, C. B. Hereditary Fragility of Bone. *Eugenics Record Office, Bulletin No.* 14, 1915, Cold Spring Harbor, N. Y.

6. Eddowes, A. Dark Sclerotics and Fragilitas Ossium. *Brit. Med. Journ., 2:222,* 1900.

7. Gates, R. Ruggles. Heredity in Man. The Macmillan Co., New York, 1929.

8. Glover, Donald M. Osteopsathyrosis: Report of a Case. *Archives of Surgery,* November, 1922, Vol. 5, No. 3.

9. Gordon, Charles A. Osteogenes's Imperfecta Congenita. *The American Journal of Obstetrics and Gynecology,* 16:214-218, August, 1928.

10. Haas, S. L. Practice of Surgery, Edited by Dean Lewis, W. F. Prior Co., Inc., 1927, Hagerstown, Maryland.

11. Harman, N. B. A Pedigree of Five Generations of Blue Sclerotics. *The Opthalmoscope,* 1910. Vol. VIII, page 559.

12. Haas, Julius. Zur Menntnis der Osteopsathyrosis idiopathica. *Medizinische Klinik* 15:1112, November, 1919.

13. Key, J. Albert. Brittle Bones and Blue Sclera. *Archives of Surgery,* October, 1926, Vol. 13, pages 523-567.

14. Nichols, Edward Hall. Surgery, Its Principles and Practice. Edited by W. W. Keen, W. B. Saunders Co., Philadelphia, 1907.

15. Oast, Samuel P. Blue Sclerotics and Brittle Bones. Report of Occurrence in Mother and Child. *Archives of Ophthalmology,* Vol. 57, No. 3, 1928, pages 254-261.

16. Ostheimer, Maurice. Fragilitas Ossium. *J. A. M. A.,* Vol. 63, pages 1996-1999, 1914.

17. Scott, Clifton R. Osteopsathyrosis (Osteogenesis Imperfecta). *The American Journal of Roentgenology.* Vol. 12, No. 3, Pages 237-242. September, 1924.

18. Shugrue, John J; Rockwood; Reed, and Anderson, Edward W. Fragilitas Ossium and Deafness, *Archives of Internal Medicine,* Vol. 39, No. 1, pages 98-108. January, 1927.

19. Spurway, John. Hereditary Tendency to Fracture. *The British Medical Journal* 2:844. September 26, 1896.

20. Simmons, Channing C. Osteogenesis Imperfecta and Idiopathic Fragilitas Ossium. *Annals of Surgery,* Vol. 46, No. 2, Aug., 1907.

21. Stobie, Wm. The Association of Blue Sclerotics with Brittle Bones and Progressive Deafness. *The Quarterly Journal of Medicine,* Vol. 17, No. 67, pages 274-287, April, 1924.

22. Straat, H. L. Blauwe Sclerae, Fragilitas Ossium en Otosklerose. *Nedr. Tijid. V. Geneesk,* Jan. 13, 1923.

23. Willard, De Forest. The Surgery of Childhood, J. B. Lippincott Co., Philadelphia, 1910.

TASSEL OF MALE-STERILE$_3$

Figure 7

A number of distinct genetic factors produce pollen sterility in maize. The gene producing this type of male sterility is located in the same chromosome as the "crinkly plant" gene. The tassels are approximately of normal size, but the branches droop. Such male-sterile forms are of practical value in the production of "crossed corn," obviating the detasseling of the plants to be used as the seed parents.

HERITABLE CHARACTERS OF MAZIE

XXXIX—Male Sterile-3

WILLIAM H. EYSTER

THAT male sterility in maize is produced by more than one genetic factor has been shown in previously published papers of this series. According to Singleton and Jones[1] male sterile$_1$ plants have a more slender tassel than their normal sibs, and produce anthers which are never extruded and do not contain pollen when mature. Male sterile$_1$ is produced by the gene *ms*$_1$ which has its locus in chromosome V near to that of *y*.

The tassels of male sterile$_2$, described by the writer,[2] are as massive as the tassels of their normal sibs, but, like male sterile$_1$, bear spikelets which contain anthers that are never extruded and the anthers contain no pollen when mature. Studies have not been made of the microsporogenous tissue in young anthers. Male sterile$_2$ is the expression of the gene *ms*$_2$ which has its locus in chromosome I.

A culture of maize plants grown during the summer of 1929 under pedigree numbers 9787 and 9788 consisted of frequencies that indicated a linkage between crinkly plant and a male sterile which differs widely from male sterile$_2$. A large number of the normal plants of these two families were self-fertilized, and progenies from several of them were grown during the summer of 1930 with the results recorded in Tables I and II. A summary of Table I is given below* in comparison with the results expected when assuming independent inheritance and linkage with 30 per cent crossing over respectively between crinkly plant and the male sterility here being discussed.

The deviations between the observed frequencies and those expected on the basis of independent inheritance of crinkly plant and male sterile are fairly wide, while the deviations between the observed and expected frequencies on the basis of linkage with 30 per cent crossing over are much less and actually agree very closely. This indication of linkage is supported by the data listed in table II which represent the repulsion series. A summary of table II is given below† in comparison with the results expected on the basis of independent inheritance and linkage with 30 per cent crossing over respectively between crinkly plant and male sterile.

Again the deviations between the observed frequencies and those expected on the basis of independent inheritance are wide, while the observed frequencies are in close agreement with the expected frequencies on the basis of linkage with 30 per cent crossing over.

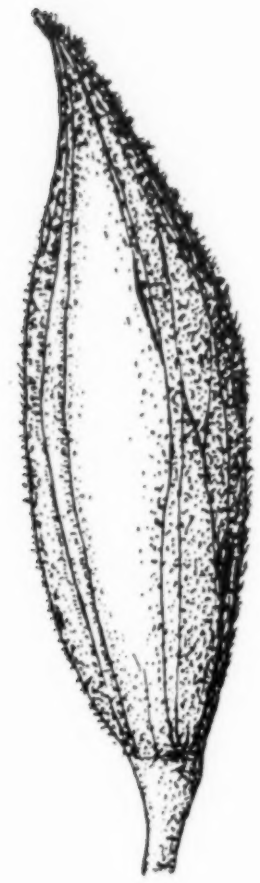
This linkage is further confirmed by a new plant character, called *yellow top,* to be described in another paper, which has been found to be linked with both crinkly plant and the male sterility here concerned.

The male sterility described in this

	Cr Ms	*Cr ms*	*cr Ms*	*cr ms*
†Observed frequencies	154	68	61	4
Expected frequencies—Independent inheritance	162	54	54	18
Expected frequencies—Linkage with 30 per cent crossing over	150	66	66	6

	Cr Ms	*Cr ms*	*cr Ms*	*cr ms*
*Observed frequencies	181	33	35	30
Expected frequencies—Independent inheritance	162	54	54	18
Expected frequencies—Linkage with 30 per cent crossing over	180	37	37	35

*Paper number 4. Contributions from the Botanical Laboratory of Bucknell University.



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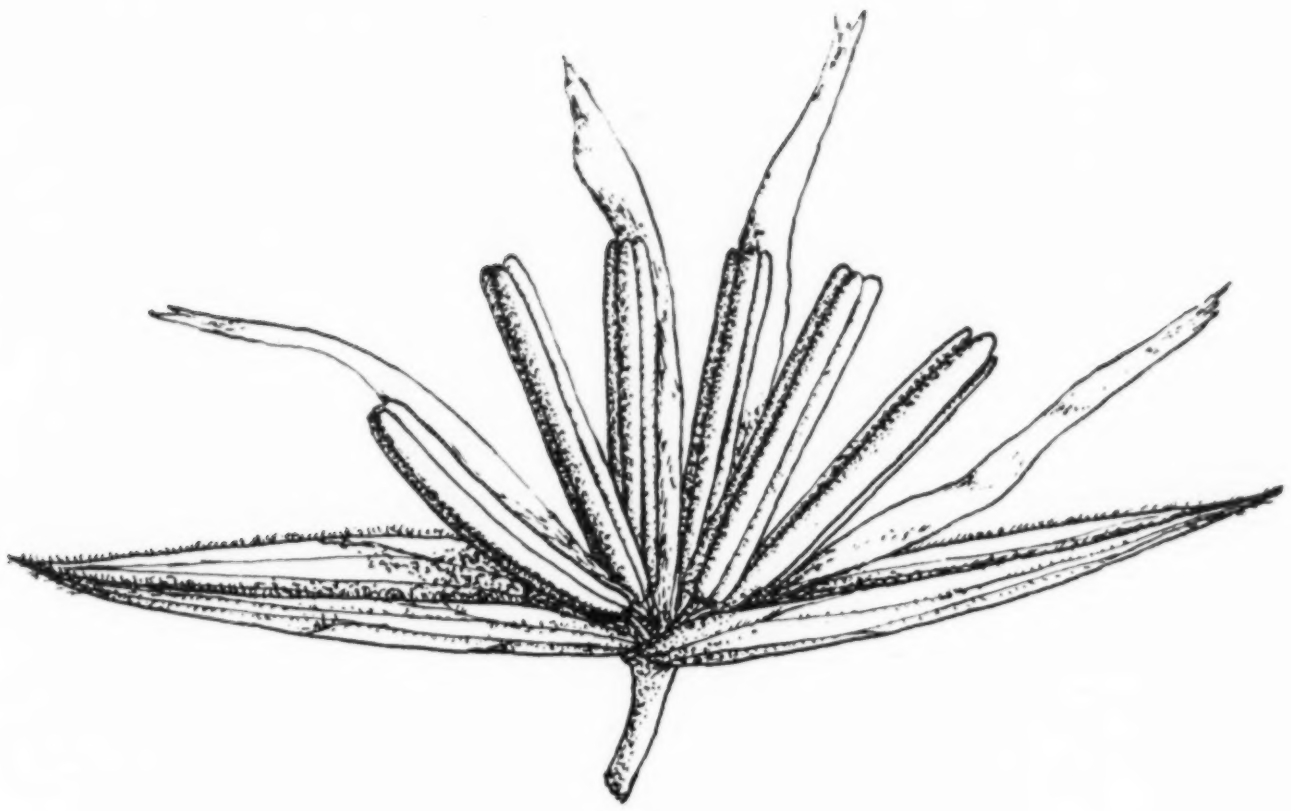

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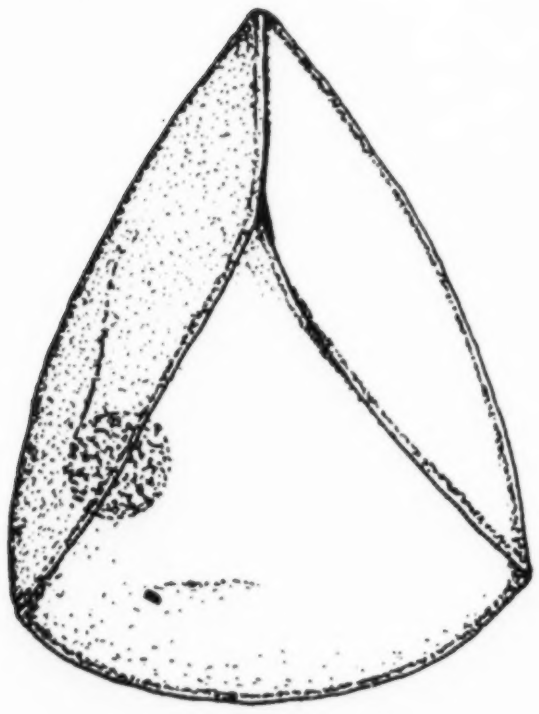

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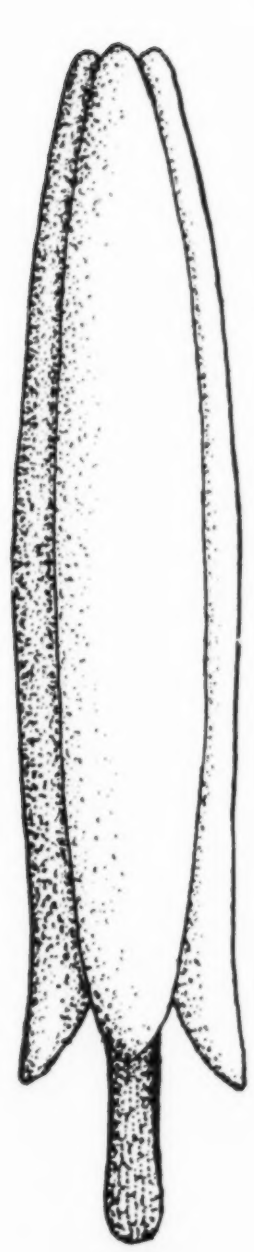

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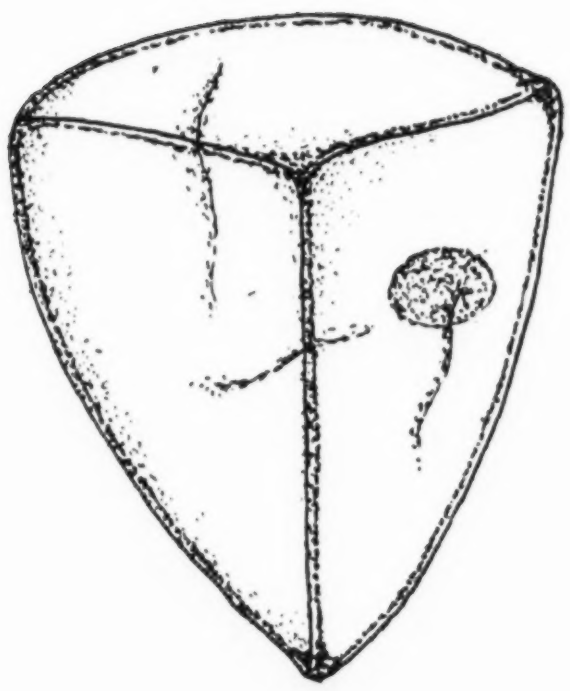

E

FLOWER DETAILS OF MALE STERILE$_3$

Figure 8

A, an unopened spikelet of male sterile$_3$. *B*, a spikelet opened to show the included stamens. *C*, an anther showing longitudinal furrowing. *D* and *E*, microspores which occur in the mature anthers of male sterile$_2$. Drawings by Miriam Groner.

paper is produced by a gene in chromosome VIII as indicated by its linkage relations with crinkly plant. This gene is designated by the symbol ms_3, and the character is called male sterile$_3$ as it is the third male sterile character to be definitely identified.

Male sterile$_3$ tassels have approximately the same size and branching as the normal fertile tassels of the same progenies. The branches, however, are more slender and are inclined to assume a drooping posture. A typical male sterile$_3$ tassel is shown in Figure 7.

Occasional spikelets of male sterile$_3$ tassels extrude anthers, though most of them do not. A typical spikelet is illustrated in Figure 8 A. Each spikelet includes six anthers as shown in Figure 8 B. The anthers are furrowed longitudinally as indicated in Figure 8 C. The anthers contain what appears upon first observation to be an abundance of pollen grains. The "pollen grains" are angular and more or less translucent, but most commonly contain only one nucleus. This would seem to indicate that the mature anthers contain microspores and not pollen. Occasionally a microspore develops into pollen as is indicated by the presence of two nuclei. Evidently microsporogenesis is accomplished but the microspores, in most cases, do not develop into male gametophytes and do not develop even so far as the pollen stage. Consequently, the mature anthers contain microspores and very few or no pollen grains.

Literature Cited

1. Singleton, R. R. and Jones, D. F. Heritable characters of maize. XXXV. Male Sterile. *Journal of Heredity* 21:266-268. 1930.

2. Eyster, William H. Heritable characters of maize, XXXVIII. Male Sterile$_2$ *Journal of Heredity* 22:99-206.

TABLE I.—F_2 progenies from the cross (Cr Ms$_3$) (Cr Ms$_3$) X (cr ms$_3$) (cr ms$_3$)

Pedigree	*Cr Ms$_3$*	*Cr ms$_3$*	*cr Ms$_3$*	*cr ms$_3$*
9788—62 (X).....	59	5	11	7
9788—64 (X).....	62	15	18	16
9788—65 (X).....	60	13	6	7
Total frequencies	181	33	35	30

TABLE II.—F_2 progenies from the cross (Cr ms$_3$) (Cr ms$_3$) X (cr Ms$_3$) (cr Ms$_3$)

Pedigree	*Cr Ms$_3$*	*Cr ms$_3$*	*cr Ms$_3$*	*cr ms$_3$*
9787—15 (X).....	130	56	54	1
9787— 4 (X).....	24	12	7	3
Total frequencies	154	68	61	4

Psychiatrist On Sterilization

TORONTO, Ontario, June 2.—Dr. Walter M. English today called upon the American Psychiatric Association, of which he is president, to approve sterilization to decrease the percentage of feeble-minded persons in the population.

Citing statistics to show that 1,800,000 persons in the United States have the mentality of children below the age of 9, he declared feeble-mindedness was increasing alarmingly in all civilized countries.

Of the 1,800,000 in the United States, he said, 600,000 are of a mental age below 7 and "these should have constant supervision throughout their lives."

He said "the great majority of cases of mental disease have one thing in common—they are the result of heredity."

Dr. English, who resides in Brockville, Ontario, spoke at the eighty-seventh annual meeting of his association.

POULTRY BREEDING AND GENETICS

At the Fourth World's Poultry Congress

THE program of the Fourth World's Poultry Congress, held in London, England, July 22-30, 1930, was so complex and varied that it is out of the question to give more than a running summary of some of the more important papers given on the poultry breeding program. Five different sessions were held consecutively each morning, these sessions being devoted to:

1. Breeding and incubation.
2. Poultry nutrition.
3. Marketing.
4. Education and extension problems, and
5. Diseases and parasites.

The sessions, as well as the entire Congress, were held at the unique glass edifice, Crystal Palace, which lent itself exceedingly well to staging the Congress.

In every respect the great avian conclove of nations was a decided success from the moment of the official opening by the Duke of York. The numerous features of the Congress contributed many things which undoubtedly will be of great value to each of the sixty countries officially represented.

Among some of the more important papers on poultry breeding are included papers from ten different countries, although only a brief discussion of some of these papers is possible here.

Professor G. S. Chlebaroff of Bulgaria, presented data on the fertilization of hens' eggs from which, very briefly, the general conclusions are: Fertility of eggs can be secured up to but not later than the nineteenth day after mating. The first egg fertilized by any male is laid on the second day after mating, irrespective of whether the last mating took place in the morning or evening. Fertility from one mating varies greatly among among hens of the same breed.

Inbreeding

Professor A. G. Dumon, Louvain University, Belgium, has found crossing inbred strains eliminates the disastrous effects of continuous close inbreeding, but serves to maintain the desirable quality bred for. It is suggested that males from inbred strains should be first tested with unrelated dams and, on the basis of results of progeny testing, used in different matings of other inbred strains.

Another contribution to the problem of inbreeding of poultry was presented by Dr. J. S. Dunkerly, Chairman of the Northern Breeding Sub-committee under the National Poutlry Institute Scheme of the Ministry of Agriculture and Fisheries. The general observations given by Dr. Dunkerly are: The production and maintenance of highly fecund stock is more likely to result from outbreeding than from either of two types of inbreeding employed, these two types of inbreeding being (1) cock mated to daughters whose ancestry was outbred; (2) cock mated to daughters derived from a brother-sister mating. In the majority of cases infertility was higher in the inbred progeny than in the outbred control. In the majority of cases father-and-daughter matings gave lower hatchability than did the control in the case of all three breeds used, White Leghorns, White Wyandottes, and Rhode Island Reds. In nearly all cases the outbred matings gave better results in the rearing of the chicks than the inbred matings. In most of the cases in the three breeds, the age of maturity was significantly higher for the inbred birds than for the outbred birds,

although there was little difference at maturity between the average weights of the inbred and the outbred progeny. The general conclusion was that it is distinctly disadvantageous to mate closely related stock.

Heterosis

Dr. D. C. Warren of the Kansas State Agricultural College, Manhattan, summarized his studies on the inheritance of vigor in the domestic fowl, from which the general conclusions are: A stimulation results from hybridization of the domestic fowl, as expressed in all stages of the life of the fowl and was measured by the hatchability of the eggs of the females outcrossed, viability and rate of growth of hybrid chicks and egg production of the hybrid females. Dr. Warren found evidence that different combinations yield varying results, and there is need for investigation to determine what combination produces the most desirable hybrids.

Hatchability and Embryonic Mortality

The writer presented a paper on the inheritance of hatchability and based his observations on hatching results in inbred strains of Barred Plymouth Rocks and White Leghorns, and non-inbred strains of White Leghorns, Barred Plymouth Rocks, and Rhode Island Reds. The results show, in general, that hatchability is inherited, but that there are also physiological factors involved that affect the result secured from any given mating. Inbreeding tends to decrease hatchability whereas outbreeding tends to increase hatchability. Moreover, as the coefficients of inbreeding increase hatchability decreases. From the standpoint of inheritance of hatchability the results suggest that possibly more than one gene or factor is involved.

Time of occurrence and probable causes of mortality in chick embryos, was the subject of a paper by Dr. T. C. Byerly of the U. S. Department of Agriculture, Washington, D. C. Briefly, Dr. Byerly found that the first peak of embryo mortality came on the third day of incubation. The first peak may be due to accumulation of excretory products and failure of the mesonephros to function. The second peak, too, may be tentatively ascribed to accumulation of excretory products since it is this period that is characterized by the relatively high combustion of protein. Oedema was frequently noted in embryos that died at this time. The usual rise in mortality during the last three days of incubation was present.

Dead-in-shell embryos were examined for malpositions as described by Sanctuary and Hutt. Over half the embyros that died during the last three days of incubation showed some malposition; 9.36 per cent of all dead embryos showed some structural malformation. These terata were classified into the usual groups; defects of eye and brain regions and ectopias were most abundant.

Thirty-two and one-tenth per cent of the eggs candled out as infertile on the seventh day of incubation were found to be fertile and to contain embryos which had died in early stages of development. Therefore, candling without subsequently breaking the eggs to check the true as opposed to the apparent fertility serves an an unreliable basis for the selection of breeding stock for fertility.

Cooperation of Genetics and Breeding

Dr. A. L. Hagedoorn, University of Leiden, Holland, discussed the different ways in which a geneticist can cooperate with poultry breeders, and among other things, suggested four lines of cooperative effort, including (a) genetical analyses of differences in characters in poultry; (b) studies involving the correlation between poultry characters; (c) studies on the effects of inbreeding; (d) experiments on the inheritance of diffrences in disease resistance; and (e)

ONE OF THE POULTRY CONGRESS EXHIBITS

Figure 9

The portion of the United States educational exhibit at the World's Poultry Congress devoted to poultry breeding problems.

experiments designed to study the inheritance of differences in fecundity, hatchability of eggs, and other factors of economic importance.

In respect to the manner in which the geneticist can cooperate with the practical poultry breeders, Dr. Hagedoorn offers the following observations:

A. Experiments for the development of new breeds.
B. Establishment of a new recessive variety of the old breed.
C. Experiments with hybrids and other progeny.
D. Teaching genetics to breeders and others associated with the poultry industry.
E. The establishment of a consultation bureau for applied genetics.
F. Cooperation with the officials of egg laying contests, concerning the development of improved systems of conducting egg laying contests.

Prof. Ch. Voitellier of the National Agricultural Institute, Paris, presented a paper on the possibilities with regard to introducing various kinds of periodical testing of egg production in place of trapnest testing. The general conclusions in Dr. Voitellier's paper are that it is quite practical to trapnest laying stock once a week, or for two or three days once a month, and the breeding stock may be selected on the basis of results from this periodical method of testing.

The relations between pedigree breeding and general utility breeding in Germany, were the subject of a paper by Professor A. Schachtzabel of the Department of Agriculture, Berlin, Germany. He believes that pedigree breeding will continue to be of the utmost importance to the German poultry industry because it will provide the basis for the distribution of

breeding stock to the utility breeders in all parts of the country.

Mr. Tom Barron, poultry breeder of Great Britain, outlined the method of conducting the progressive breeding test known as the Lancashire breeding scheme. The pullets of certain breeds at the different laying trials must lay at least 200 first-grade eggs, and in other breeds pullets must lay at least 180 first-grade eggs during the time of the official contest which lasts 48 weeks. The first four weeks of the official tests the eggs weighing 1⅞ ounces each are graded as first-grade eggs. From the fifth to the eighth week, the eggs must weigh 1-15/16 ounces each; and from the ninth to the forty-eighth week eggs must weigh two ounces each in order to grade as first-grade eggs. Hens at the different laying trials which qualify on the basis of graded eggs, as mentioned above, may then be sent to the breeding station where they are mated to males supplied by the owner of the females. Cockerels are required to pass inspection of the Committee as to type, pedigree, and other qualifications. The chicks are pedigree-hatched, so that the buying public may purchase pedigreed chicks of known ancestry.

Dr. Alula Taibell, Robigo, Italy, gave a paper on the selection of egg-laying stock from the crossing of pure Leghorn stock with local Italian breeds. His general conclusions are that the use of pedigreed White Leghorn males on ordinary Italian hens serves to increase egg production very materially particularly with respect to fall and winter egg production. Dr. Taibell suggested that the purebred White Leghorn males used in the matings were heterozygous for high egg production inasmuch as the records of progeny were about midway between the records of purebred White Leghorn hens and those of ordinary Italian hens.

Dr. Kazunobu Kimura of the Imperial Zootechnical Experiment Station, Chiba-shi, Japan, gave an interesting paper on pedigree breeding for egg production with a flock of White Leghorns. The data presented covered breeding records from 1919 to 1929, inclusive. Egg production was increased from an average of 142.50 during the laying year 1919-1920, to 230.16 during the laying year 1928-1929. During the same period the average weight of eggs in grams remained practically the same. One of the most important factors affecting the increase in annual egg production was the earliness with which the pullets came into egg-laying condition, the average number of days from hatching time to egg laying being 229.58 days in 1919-1920, and 180.00 in 1928-1929. Dr. Kimura also gave some information regarding the number of 300-egg hens secured in the Mikawa, a native breed.

Dr. Walter Landauer, Storrs Agricultural College, Connecticut, discussed the "Genetics and Biology of the Creeper (Scotch Dumpie) and the Frizzle Fowl." He states that a comparison of the results of his investigations concerning the Creeper and the Frizzle fowl shows interesting similarities and differences between the two breeds. In both cases the character is determined by a single Mendelian dominant. The Creeper character is lethal in homozygous condition, the Frizzle character is not lethal. In the Frizzle, however, the homozygous individuals show a decrease in viability. In *inter se* matings of both Creepers and of Frizzles (of exhibition type) the embryonic mortality is high and the hatchability consequently low. In the case of Creepers this is accounted for by the death of the homozygous embryos (25 per cent of all fertile eggs), while in the Frizzle the excessive embryonic mortality is not genetic *sensu stricto* but must be explained by the action of physiological factors. On account of their physiological association with these particular characteristics the

detrimental traits of both Creeper and Frizzle fowl cannot be eliminated but must remain apart of the price the fancier has to pay for his desire to maintain these breeds.

Dr. Frank A. Hays of the Massachussetts Agricultural Experiment Station, Amherst, presented his general conclusions regarding the linkage relations between genes for egg size and genes concerned in high fecundity in the domestic fowl. He found that no linkage relations appear between genes *A* and *B* for egg size and genes *E* and E^1 for early sexual maturity. No linkage was observed between genes *A* and *B* for egg size and gene *M* for winter pause. Gene *B* for large eggs and gene *I* for clutch size from 2.1 to 3.0 are linked and gave a crossover value of about 34.04 per cent. Gene *A* for small eggs is linked with gene I^1 for clutch size above 3.0. The crossover value is about 36.56 per cent. There is apparently no linkage between the two genes for egg size and the two genes for broodiness. The data do not suggest linkage between genes for egg size and gene *P* for high persistency.

Other Papers

Many other papers on breeding and incubation were given in the sessions devoted to these two subjects but lack of space prevents even a brief review.

United States of America, 1936?

The First World's Poultry Congress was held at the Hague in 1921; the second was held at Barcelona, Spain, in 1924, the third was held at Ottawa, Canada, in 1927, and the fourth was held in London, England, in 1930, and the fifth will be held in Rome in 1933. Practically everyone has agreed, therefore, that the sixth Congress should be held in the United States in 1936. Judging from the benefits accruing to the poultry industry in the countries in which the congresses have been held, there can be no question but that practically all the poultry organizations in the United States would be very desirous of seeing the 1936 congress held in the United States. As a matter of fact, one or two cities have already put forth efforts to see if arrangements could not be made to have the congress held in their cities.

The congresses are of very decided advantage to the countries in which they are held, and in the United States the Sixth World's Poultry Congress would be an excellent stimulus for increased activities and further efforts on the part of such organizations as the International Baby Chick Association; the United States Egg Society; the National Poultry, Butter and Egg Association; the National Poultry Council; the Standardbred Poultry Association of America; the Poultry Science Association, and other organizations. Undoubtedly all State institutions and departments of agriculture would be greatly interested in the possibility of holding the Congress in the United States in 1936. The final decision, however, as to where the congress is held rests with the Executive Council of the World's Poultry Science Association.

MORLEY A. JULL.

THE INHERITANCE OF "HAIRLESSNESS" IN SWINE

Hypotrichosis II*

ELMER ROBERTS AND W. E. CARROLL
University of Illinois

MEXICAN hairless swine have been known for some time in certain parts of Mexico. This character is not associated with any other abnormal condition and while apparently hereditary, its mode of inheritance has not been known previous to the present study, the object of which was to obtain information on this point.

This type of hypotrichosis or hairlessness is entirely distinct from the hairlessness prevalent in certain regions of the United States, which is caused by a deficiency of iodine probably associated with goitre in the mother. In the latter kind the young are either born dead or die within a few hours after birth. The administration of iodine will prevent the occurrence of this type, but the hairlessness of the Mexican swine can not be prevented in this way. The first type of "hairlessness" has been studied and reported by several workers, among whom are Welch[8], Smith[7], and Hart and Steenbock.[1]

The term "hairlessness" is in reality a misnomer, since all of the so-called hairless individuals which have been observed possessed some hair; hence the term hypotrichosis (congenital deficiency of hair) is better, though "hairless" is the term in common use. Hypotrichosis has been used by one of the authors, Roberts,[2,3] and by Roberts and Thomas[4] in recent publications dealing with hair abnormalities in the rat and in man.

For the present study, hypotrichotic swine, two males and two females, were imported from Ario, Michoacan, Mexico. One of these, a female, proved to be heterozygous, leaving two males and one female homozygous for the hairless condition. Figures 10 and 11 show the difference between a Mexican hairless and a normal-haired individual. The heterozygous Mexican female (Figure 10) had a smaller amount of hair than had normal swine, but considerably more hair than the "hairless" Mexicans. Progeny from a Mexican hairless male mated to grade Chester White females showed the same condition of hair, intermediate in amount between the normal and hairless states. So the normal condition which we shall designate *H* is incompletely dominant to the "hairless" condition designated by *h*. Sixteen F_1 individuals, seven males and nine females, were produced, all of which presented this intermediate condition.

Figures 12 and 13 are profile pictures of pieces of skin from normal-haired, heterozygous, and hairless individuals, respectively. For these exposures the skins were placed around a cylinder in order to show better the distribution of hairs. The differences are strikingly apparent. Figure 14 is a view of flat surfaces of similar pieces of skins, showing in general the same differences.

Sections of skin were taken from animals of the various genetic constitution, after being dressed for market, and photographed in order to show the hair pores. Figure 15 shows the relative numbers of hairs represented

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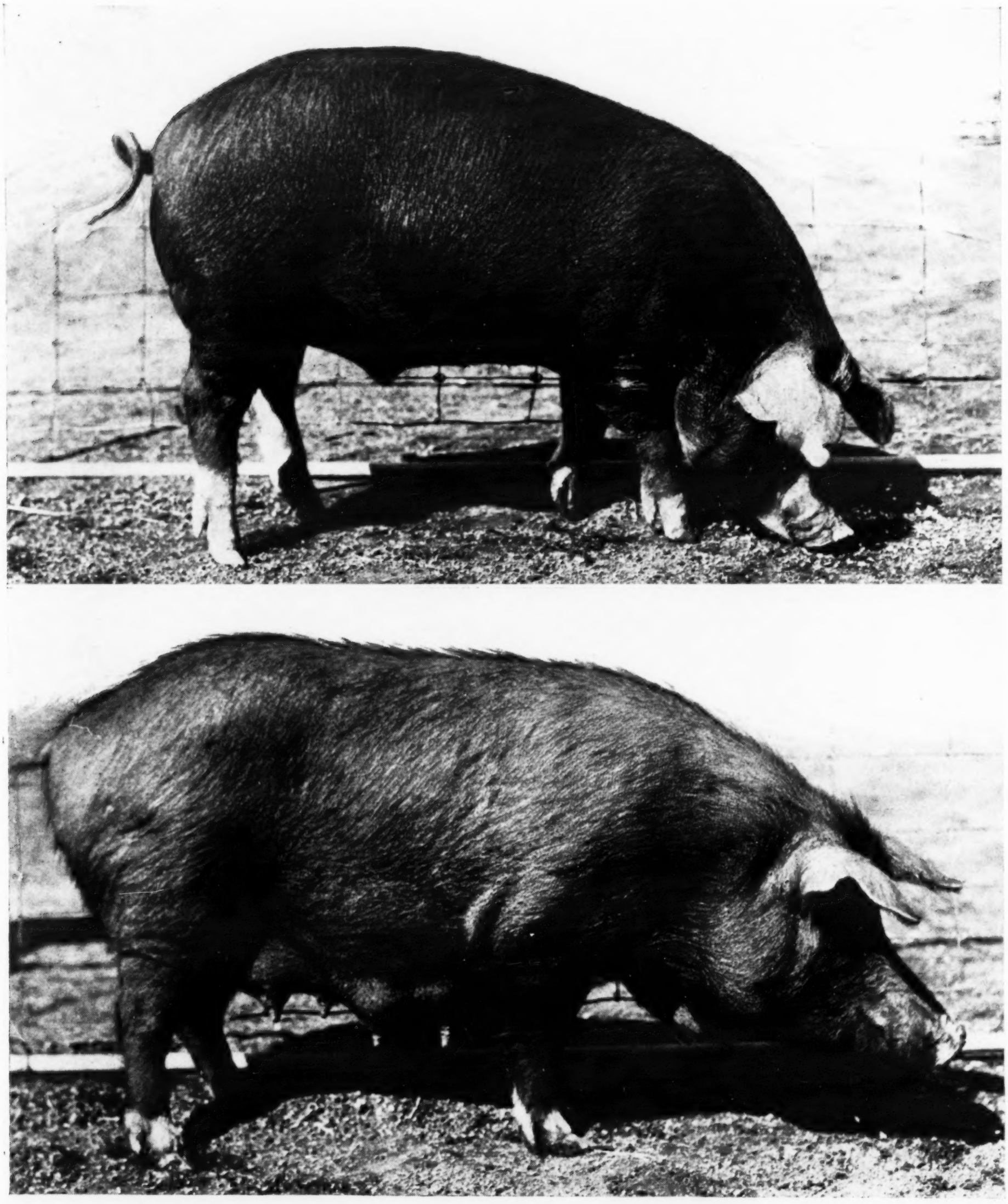
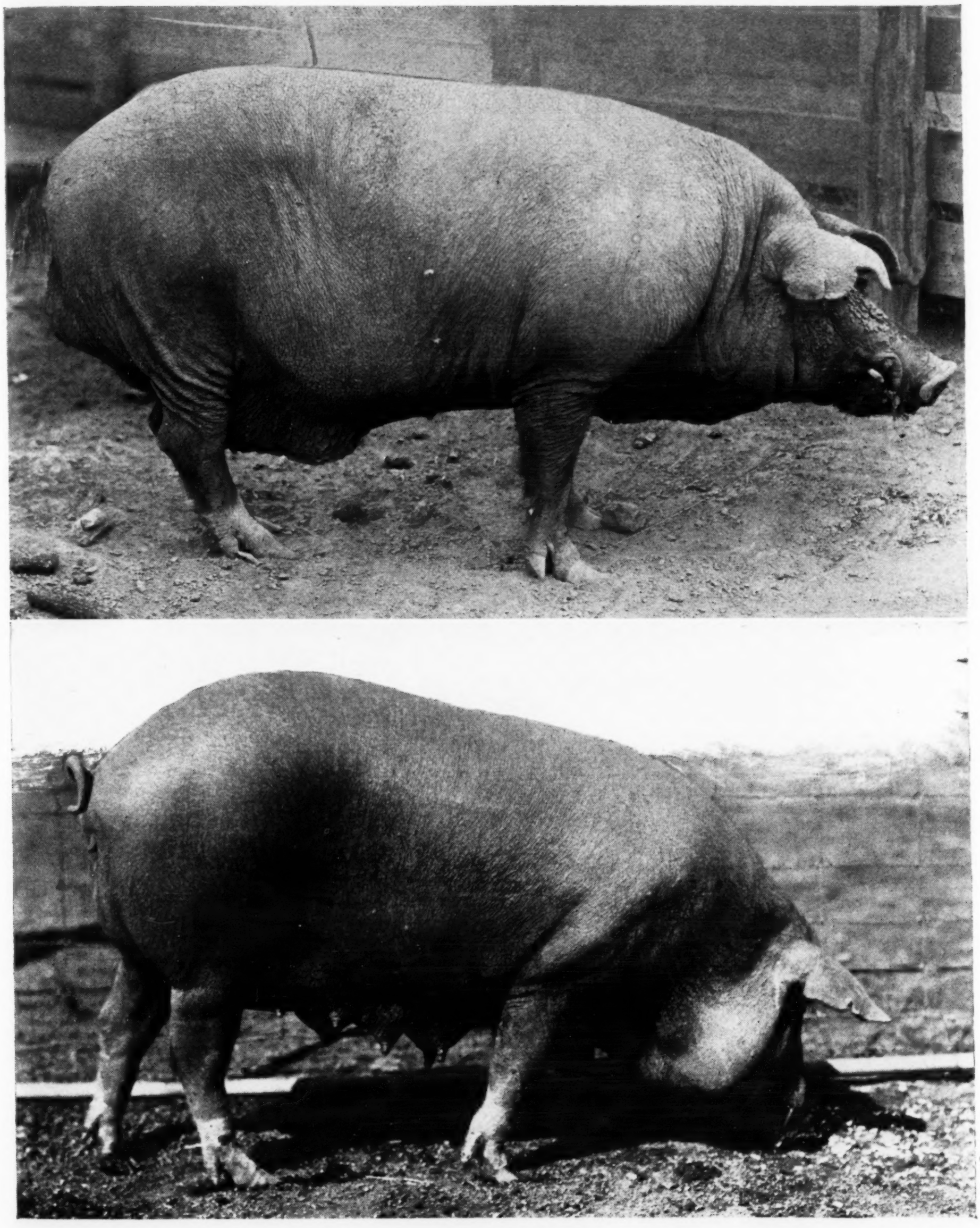
*Paper No. 40 from the Division of Animal Genetics in cooperation with the Swine Division, Department of Animal Husbandry. The first paper of the series was published in *Eugenics*.[4]



HAIRED AND "HALF-HAIRED" PIGS

Figure 10

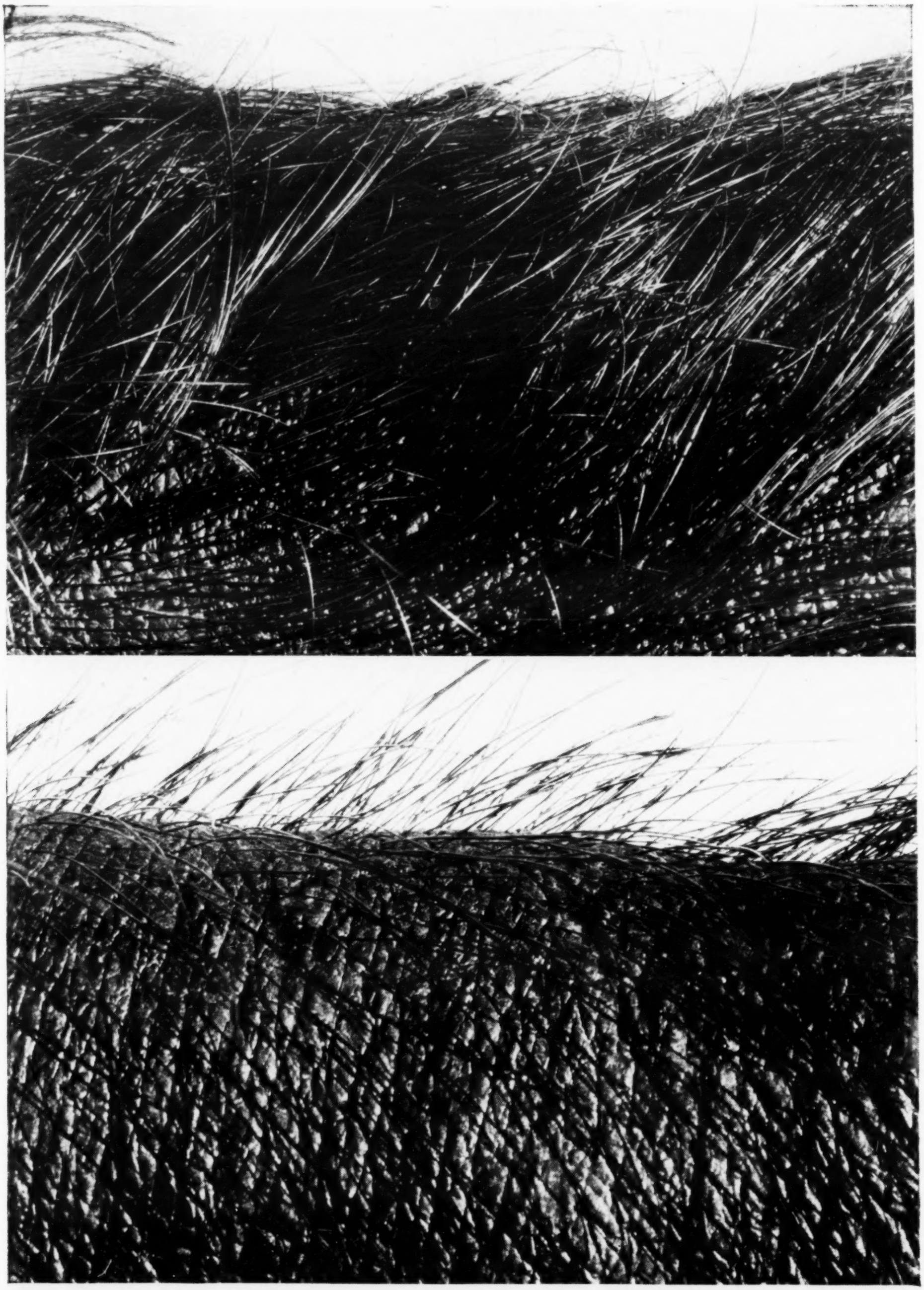
Above, a normal Poland China boar (*HH*); below, a sow imported from Mexico, that is heretozygous for hairlessness (*Hh*). She has considerably less hair than normal.



"HAIRLESS" PIGS

Figure 11

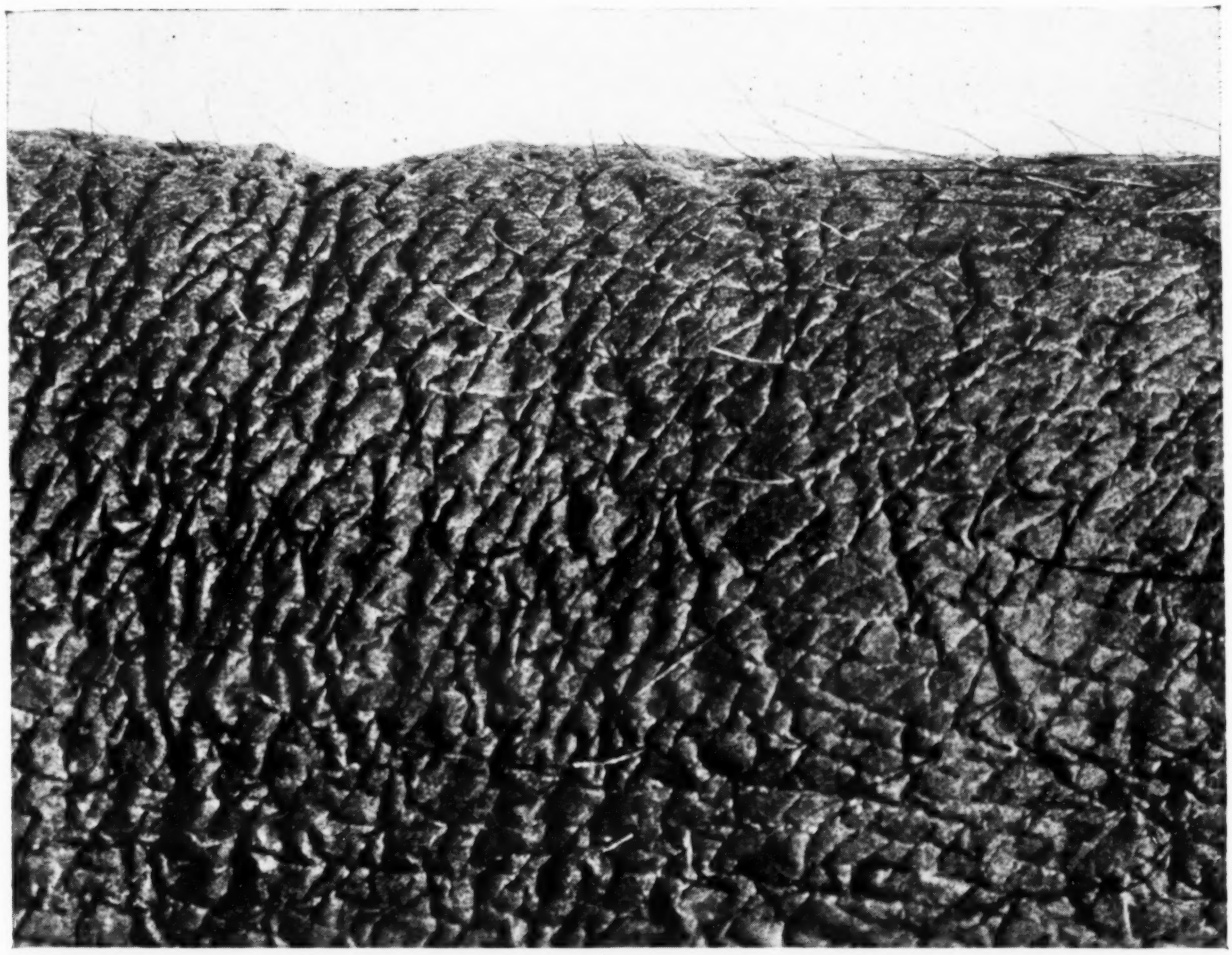
Above, a hairless boar from Mexico, and below, a hairless segregate from the mating of a heterozygous (*Hh*) Mexican sow with a hairless (*hh*) Mexican boar. Hairlessness is inherited as a Mendelian character.



HAIRLESSNESS PARTIALLY DOMINANT

Figure 12

Above is shown a section of rolled skin of a normal Poland China pig; below, that of a pig heterozygous for hairlessness (*Hh*). The heterozygous animals have about half as much hair as an ordinary pig.



A "HAIRLESS" SKIN

Figure 13

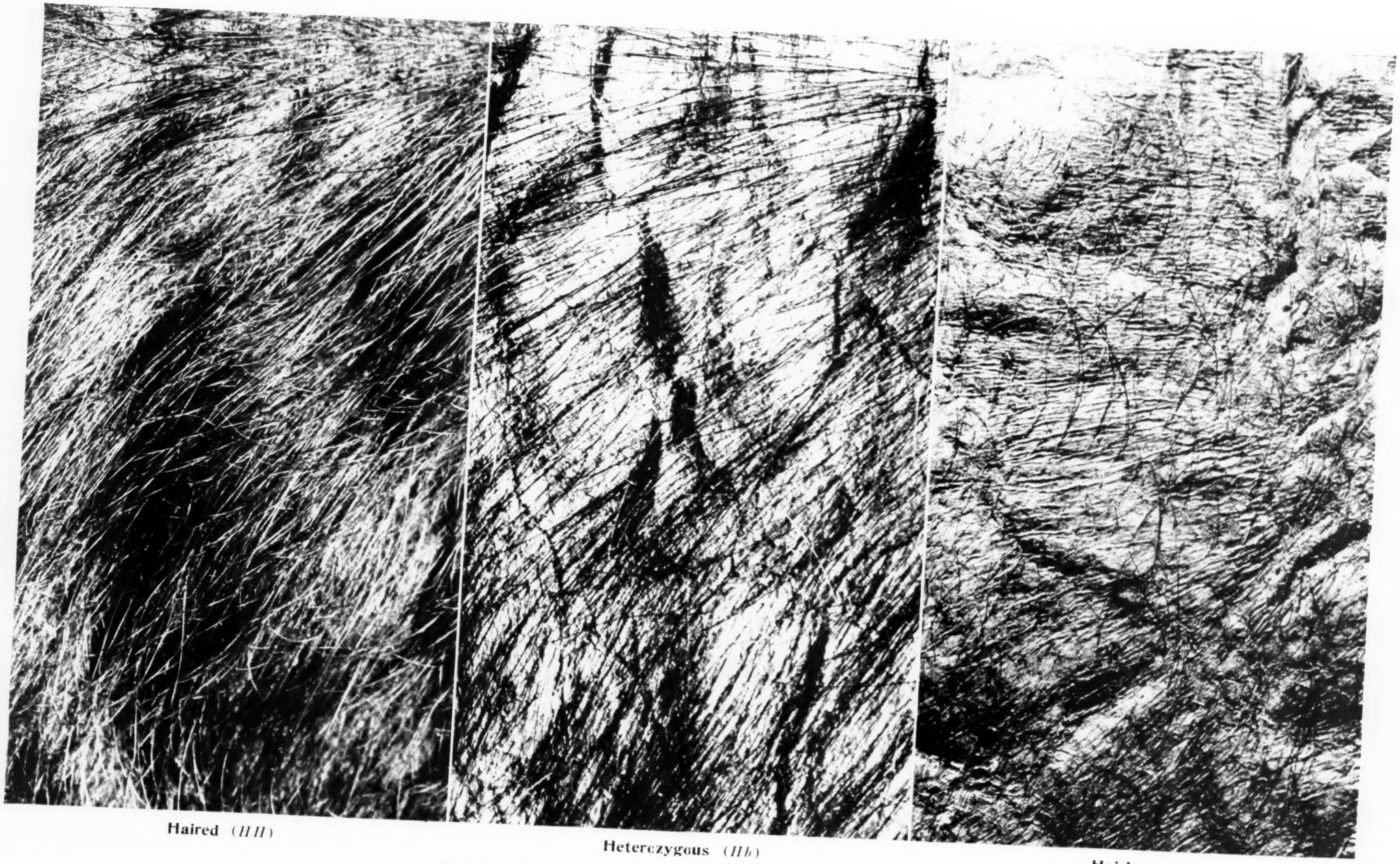
The skin of the hairless pig has a sparse covering of hair. Another form of hairlessness in pigs, induced by a lack of iodine in the diet of the mother suggests that the hereditary form of hairlessness might have a thyroid abnormality as its basis. This appears not to be the case, because iodine treatment has no effect, and because the pigs are otherwise normal. The iodine-deficient hairless pigs are born dead or survive only a few hours after they are born.

by the pores. In Figure 15 the pores of the " hairless" skin are much larger than in either of the other cases, which may be due to the much greater age of this animal, one of the animals obtained in Mexico in 1926 and at least five years old at the time the section of skin was taken. The other animals were under two years of age.

Preliminary notes concerning the inheritance of this hypotrichotic or "hairless" character have been published in the annual reports [5,6] of the Illinois Agricultural Experiment Station. Since then, more data have been secured, and the present publication contains all results available to date.

Matings of two normal females and a Mexican hairless boar produced sixteen young, seven males and nine females. Three of these F_1 females together with the heterozygous Mexican female were mated with the Mexican hairless males. From these matings 53 young were produced, 28 of which were like the Mexican hairless and 25 were haired like the hybrids, or heterozygous individuals. The offspring of hairless $\times$ hairless were like the parents.

These results indicate that the difference between the normal and the



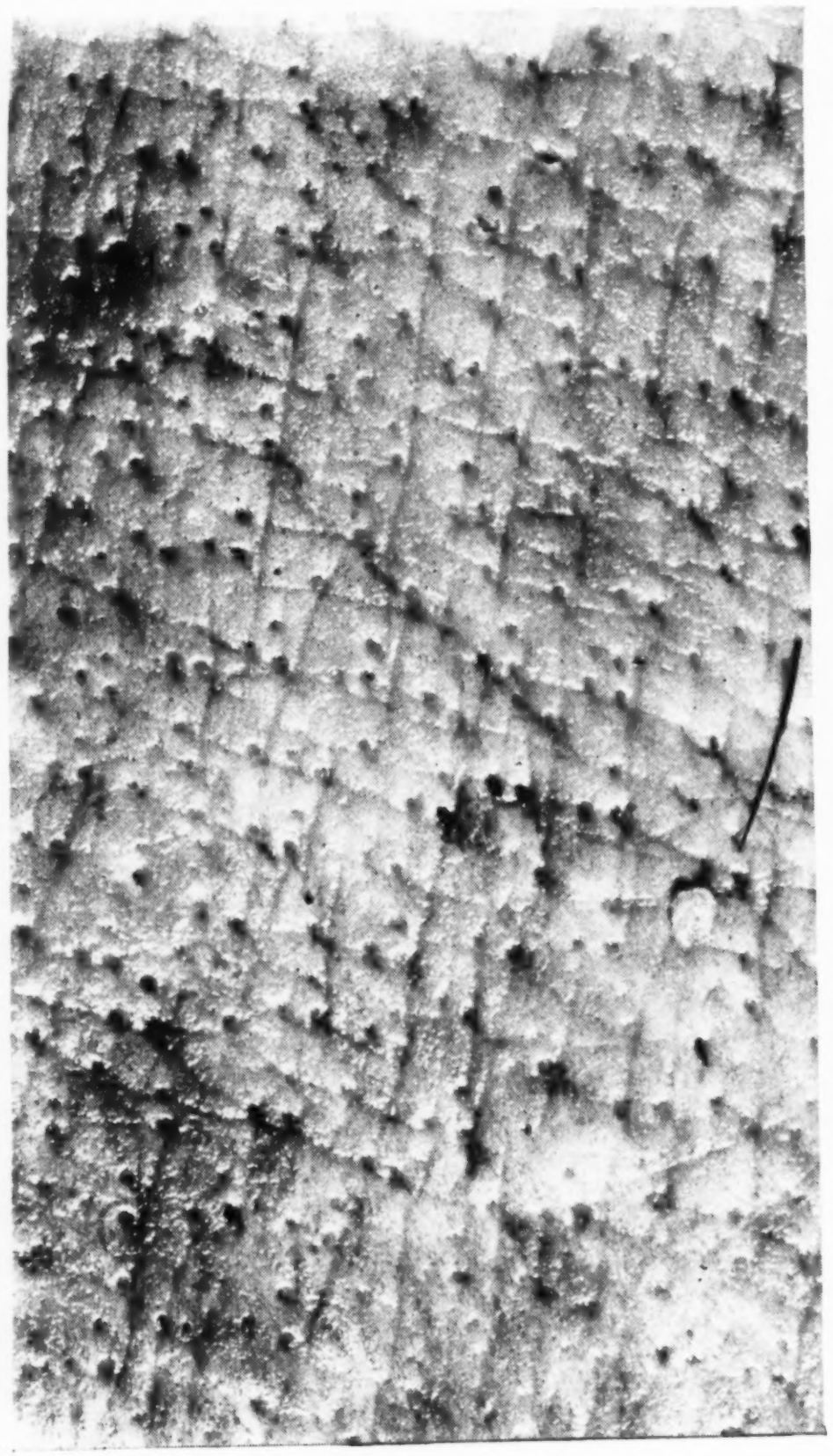
Haired (*HH*) Heterozygous (*Hh*) Hairless (*hh*)

VARYING DEGREES OF "HAIRLESSNESS"

Figure 14

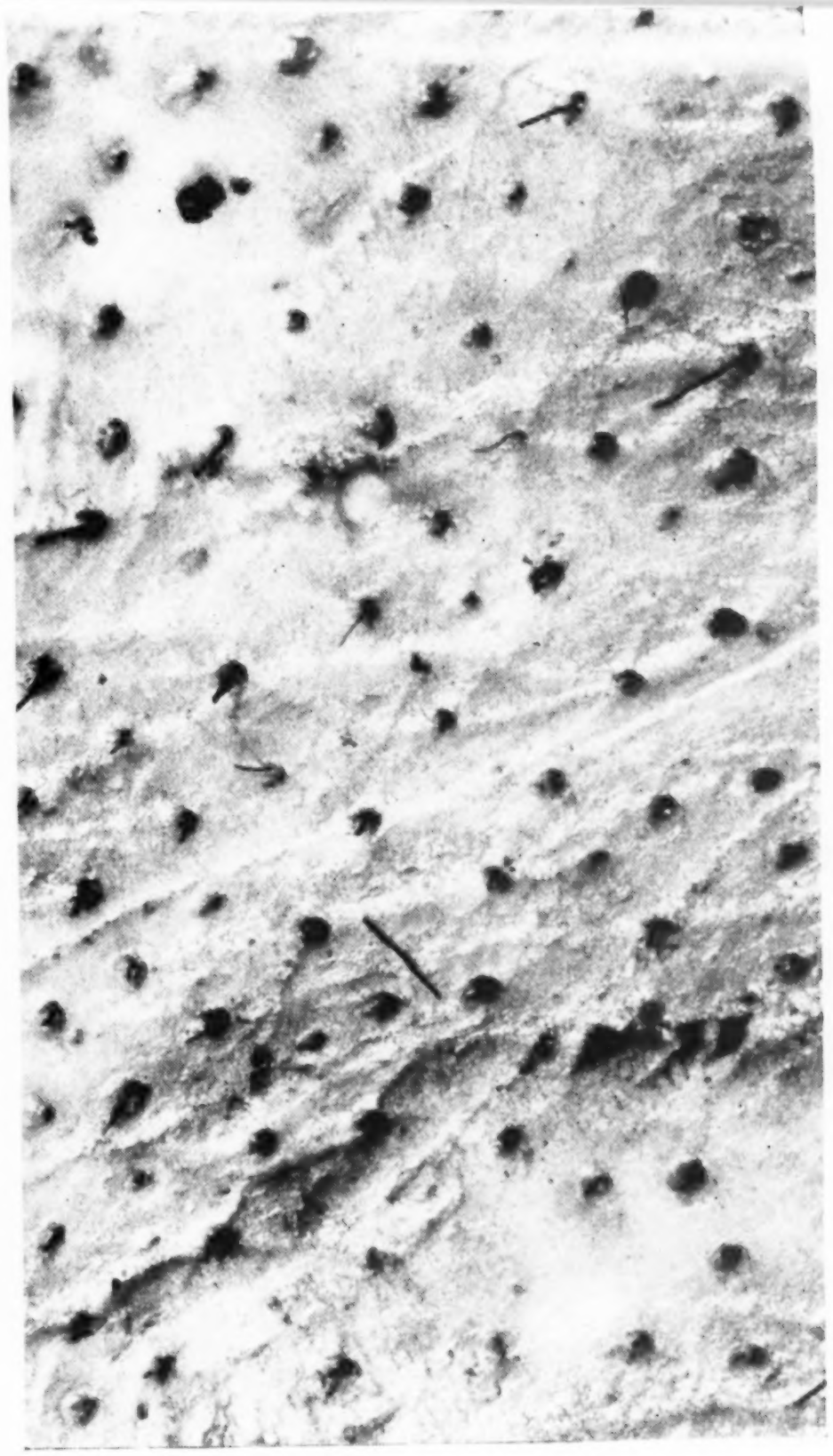
"Bird's-eye view" of skins of the three hair-types, showing the intermediate amount of hair on the heterozygous skins.

the heterozygous skins.



Haired

Heterozygous



Hairless

DISTRIBUTION OF HAIR FOLLICLES

Figure 15

Sections of shaved skin to show the distribution of the hair follicles in the three classes. The larger pores of the hairless (*hh*) skin are probably due to the greater age of the animal. A histological examination of the three types shows that the follicles, though much less numerous in the hairless skin, are nevertheless normal in structure.

"hairless" condition is due to a single factor or gene. However, the normal is only partially dominant as shown by examination of the various illustrations. Table 1 gives the results of the various kinds of matings. The symbols used are H = normal condition, and h = the hypotrichotic or hairless condition.

Table I. Matings involving normal (H) and hairless (h) genes.

Mating	*Haired (Hh)* ♂	♀	*Hairless (hh)* ♂	♀
HH × *hh*	7	9	....	....
Hh × *hh*	15	10	19	9
hh × *hh*	....	....	9	4

While the amount of hair on the homozygous hypotrichotic individuals varies, yet there is no overlapping with the heterozygous forms.

The cause of the hair abnormality under discussion is not known, but it is not a deficiency of iodine unless there is an inherited defect of the thyroid glands. If this were true, abnormal pigs would be farrowed. In the case of hairless rats, the thyroids are normal and the administration of iodine and cystine failed to promote hair growth. Some of our "hairless" pigs weighing 40-50 pounds have been fed 0.5 to 1.0 gram of cystine daily for as long as twenty-eight days without noticeable effects.

Through the kindness of Dr. Frank Thorp of the Laboratory of Animal Pathology and Hygiene of the University of Illinois, histological studies were made of the hair follicles of sections of skin from the various kinds of animals. While the number of follicles was much smaller in the hairless and heterozygous individuals than in the homozygous haired, they were normal, no pathological conditions of any kind being observed.

Literature Cited

1. Hart, E. B. and H. Steenbock, 1918. Hairless pigs, the cause and remedy. *Wisconsin Agric. Exp. Sta. Bull.* No. 297.

2. Roberts, E. 1924. Inheritance of hypotrichosis in rats. *Anat. Record,* vol. 29, p. 141.

3. Roberts, E. 1926. Further data on inheritance of hypotrichosis in rats. *Anat. Record,* vol.. 34, p. 172.

4. Roberts, E. and L. C. Thomas, 1930. Hypotrichosis: The inheritance of monilethrix. *Eugenics,* vol. 3, pp. 20-25.

5. Roberts, E. 1927-1928. *Annual Report of Ill. Agr. Expt. Sta.* pp. 124-25.

6. Roberts, E. 1928-1929. *Annual Report of Ill. Agr. Expt. Sta.,* p. 82.

7. Smith, G. Ennis (with cooperation of *Howard Welch*) 1917. Fetal Athayrosis. *Jour. Biol. Chem.* Vol. 29, pp. 215-225.

8. Welch, Howard, 1917. Hairlessness and goitre in new-born domestic animals *Montana Agric. Exp. Sta. Bull.* No. 119.

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COLOR CHIMERAS IN THE DOMESTIC FOWL

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A GREEN AND A YELLOW LEG

Figure 16

A chimeral fowl which has legs of different colors, the darker leg being green and the lighter yellow. Such a condition might be produced either by unequal chromosome distribution in the two sides (non-disjunction) or by gene mutation.

WITHIN recent years several unusual color mutations have been noticed by the author and have also been reported in the literature. Pigment mutations in poultry seem not to be uncommon.

A previous report on a "half and half" skin-color mosiac in the fowl by Lambert[3] of this station has already been made. In addition to this, Lambert and the writer have noted two pigment mutations in the F_1 progeny from a cross of Black Langshans with White Plymouth Rocks. One of these was a "half and half" skin-color mosaic similar to the one reported by Lambert[3]. Another had both shanks white in color but one shank had a yellow streak. Tentatively we may suppose that these were due to somatic gene mutations or non-disjunction.

Recently a cross-bred female at the college farm was found to have one

green and one yellow shank. The right shank was green and appears as the darker of the two shanks in Figure 16. She was an F_1 female sired by a Rhode Island Red, which was mated to a White Wyandotte female.

According to Barrows[1] the pigmentation of the skin of the bird is due to the presence of pigment either in one or both layers of the epidermis of the shanks. He found that there were two classes of pigments responsible for the color of the shanks. The zooxanthin (yellow) pigment, in both layers of the epidermis or in the corium alone, produces yellow shanks, while a lack of this pigment produces white shanks. The other pigment is melanin which causes a variety of darker coloration of the shanks. This variation of color is augmented by the presence or absence of the lipochrome pigment. The apparent depth of color of the melanic pigment is dependent upon whether or not the melanin is deposited in the chorium, malphigian, or dermal cells. The nearer the surface that the pigment is deposited, the darker is the color of the shank. The darker hues also have the melanic pigment deposited in all layers of cells. In this particular instance, the left shank has no melanic pigment but only the lipochrome, which makes the shank appear yellow. The right shank has the lipochrome color plus the deposition of the melanic pigment in the upper chorium, producing a greenish effect. This was ascertained by Dr. H. E. Biester who made a histological examination of the shank tissues of the mutant. This agrees with Barrows[1].

There are two possible explanations for this phenomenon. One is that it was caused by non-disjunction of the chromosomes carrying one of the major genes for melanic pigment. The other is that it was caused by a mutation of the gene determining the production of melanic pigment.

If this condition were due to non-disjunction, it would naturally be expected that other characters would be affected besides that of pigmentation, but nothing of this kind was observed. Hutt[2] in the case of the mosiac reported by Lambert[3] states that were the mosiac caused by non-disjunction the white side of the fowl would probably be larger. No such diversity of size was apparent in this particular case. On the other hand, the domestic fowl has so many pairs of chromosomes that the misbehavior of one pair might not be readily apparent, especially if there were many chromosomes involved in determining the character as is probably the case of the inheritance of size. The writer is inclined to believe this to be a gene mutation. Although both birds of the parental cross had yellow shanks, it is fairly common to find White Wyandottes having green shanks, and many of the yellow shanked birds produce some progeny with green shanks. In this instance it is believed that the mutation is due to a loss or change of the gene producing the melanic pigment in a bird that would normally have been a green-shanked bird. It also could have been a dominant mutation producing one green shank while the other retained the recessive color, in this case yellow.

Literature Cited

1. Barrows, 1914. Histological basis of shank colors in domestic fowl. *Me. Agr. Exp. Sta. Bull.* 232.

2. Hutt, F. B., 1929. A note on Lambert's mosaic in the fowl. *Jour. of Heredity* 20:323-324.

3. Lambert, W. V., 1929. A "half and half" skin-color mosaic in the chicken. *Journal of Heredity* 20:167-169.